ISSN 0972-3757
INTERNATIONAL JOURNAL OF HUMAN GENETICS
Journals ON-LINE Publishers Since 1933

Int J Hum Genet, 9(2): 131-137 (2009)
PRINT: ISSN 0972-3757 ONLINE: 2456-6360
DOI: 10.31901/24566330.2009/09.02.08

Genetic Markers in Idiopathic Pulmonary Arterial Hypertension (IPAH)

Sujana Katta*, Shivani Vadapalli*, R.Sushant, B. K. S. Sastry*** and Pratibha Nallari ***

Department of Genetics, University College of Science, Osmania University, Hyderabad, Andhra Pradesh. India
****Kakatiya Medical College, Warangal, Andhra Pradesh, India***
*****Cardiology Unit, CARE Hospital, Hyderabad, Andhra Pradesh, India***

KEYWORDS Idiopathic Pulmonary Arterial Hypertension. Superoxide Dismutase. Catalase. α-1-Antitrypsin. Alpha-1- Antitrypsin

ABSTRACT Idiopathic pulmonary arterial hypertension (IPAH) is a rare disorder with abnormally raised pulmonary arterial pressure. In IPAH, the trigger of the endothelial injury may result from oxidative stress, hypoxia, shear stress, inflammation in conjunction with genetic susceptibility. These epigenetic factors may have an influence on cell growth, differentiation and normal homeostatic functions of the endothelium, by altering endothelial permeability, production of growth factors and coagulation factors. Lung inflammation can lead to increased levels of oxidants that may also contribute to the development of IPAH. When the production of ROS exceeds, the capacity of the cell to detoxify them decreases and the resulting oxidative stress may be harmful to the integrity of biological tissue. Since in IPAH there is disruption of pulmonary artery vasculature, the generation of free radicals may further aggravate tissue injury and in view of its role in defensive mechanism, qualitative variation of SOD, CAT, AAT in IPAH is investigated in the present study to correlate specific electromorphic associations in its etiopathogenesis. In conclusion the present study revealed that Oxidative stress pathway (ROS/RNS) and inflammatory components may act as modifiers in pathogenicity of IPAH.